

# CONTROLLING WILDLIFE AND LIVESTOCK DISEASE WITH ENDOGENOUS ON-FARM BIOSECURITY

Richard D. Horan, Christopher A. Wolf, Eli P. Fenichel, and Kenneth H. Mathews, Jr.

[horan@msu.edu](mailto:horan@msu.edu) & [wolfch@msu.edu](mailto:wolfch@msu.edu)

Department of Agricultural Economics  
Agriculture Hall  
Michigan State University  
East Lansing, MI 48824-1039

## Abstract

The spread of infectious disease among and between wild and domesticated animals has become a major problem worldwide. We analyze the socially optimal management of wildlife and livestock, including choices involving environmental habitat variables and on-farm biosecurity controls, when wildlife and livestock can spread an infectious disease to each other. The model is applied to the problem of bovine tuberculosis among Michigan white-tailed deer. The optimum is a cycle in which the disease remains endemic in the wildlife, but in which the cattle herd is depleted when the prevalence rate in deer grows too large.

*Paper prepared for presentation at the American Agricultural Economics Association  
Annual Meeting, Denver, Colorado, August 2004*

\*Copyright 2003 by Richard D. Horan and Christopher A. Wolf. All rights reserved. Readers may make verbatim copies of this document for non-commercial purposes by any means, provided that this copyright notice appears on all such copies. The authors gratefully acknowledge funding provided by the Economic Research Service-USDA cooperative agreement number 43-3AEM-3-80105 through ERS' Program of Research on the Economics of Invasive Species Management (PREISM). The views expressed here are the authors and should not be attributed to ERS or USDA. Thanks to Erwin Bulte, Larry Karp, and Aart de Zeeuw for helpful comments and suggestions. The usual disclaimer applies.

## **Introduction**

The spread of infectious disease among and between wild and domesticated animals has become a major problem worldwide. Most policy responses involve extreme reactive measures such as attempts to eradicate all wildlife in an infected zone and depopulating infected livestock herds. A primary motivation for such measures is to protect livestock industries. The wildlife-related benefits from undertaking alternative disease control investments may be poorly understood. It is also not clear how much weight is given to the public good nature of on-farm biosecurity measures that prevents the spread of the disease.

The economic literature has primarily followed the trend set by policy makers, with most animal disease work providing estimates of the costs to farmers and consumers under alternative control strategies, with little regard given to the wildlife dimension (e.g., Mahul and Gohin 1999; Kuchler and Hamm 2000; McInerney 1996; Ebel et al., 1992; Dietrich et al. 1987; Liu 1979). While there has been relatively little research in the area of the economics of disease control among wildlife populations, a wildlife disease outbreak may impose significant costs on those who value wildlife products and/or services. For instance, an infectious wildlife disease might impose costs on hunters who place a premium on healthy wildlife. Costs may also arise as infected or even healthy populations in close proximity to an outbreak are culled to prevent additional spread. The costs could be greater for threatened or endangered species, particularly those protected in parks not large enough to support a viable population. As population members wander outside protected areas, the risk of infection increases – both for wandering individuals and for those in protected areas. Conservation measures must therefore be taken with disease control in mind (Simonetti 1995).

A few studies have explicitly considered the wildlife component of animal disease. Bicknell et al. (1999) analyzed the private incentives that a New Zealand farmer would have to undertake disease control measures for the case of bovine TB, which is spread by Australian brushtailed possums to dairy herds. Bicknell et al. (1999) developed a bioeconomic model involving healthy and infected possum populations and also a dairy cow population. They then explored optimal disease control strategies for a single farmer, including testing at the farm level, and hunting possums off the farm. Possums in that model were primarily a nuisance, possessing no significant values for alternative uses. Horan and Wolf (2003) extended this line of research by considering the social planner's problem for a situation in which the wildlife held significant recreational value. They also incorporated the realistic feature that infected wildlife cannot be identified until after they are killed and examined (Williams et al. 2002), rendering it impossible to selectively harvest only infected animals. As a result of these features, they found that exterminating wildlife as a way of eradicating a disease outbreak, as is often proposed, might be a comparatively costly approach. But while Horan and Wolf (2003) did model damages to a livestock sector (through a damage function), they did not explicitly consider on-farm management choices that could affect the risk of disease transmission from wildlife to livestock and vice versa. The purpose of the present paper is to explicitly consider these choices within the context of the social planner's problem.

The model is applied to the case of bovine TB among white-tailed deer in Michigan, the only known area in North America where bovine TB has become established in a wildlife population. Bovine TB, which was responsible for more livestock deaths than all other diseases combined at the turn of last century (MDA 2002), is currently being transmitted among and between white-tailed deer and dairy cows and captive cervids in Michigan. The USDA awarded

Michigan TB accredited-free status in 1979 (MDA 2002). This important accreditation prevents other states from imposing trade restrictions on Michigan livestock and livestock products. But in the early to mid-1990s bovine TB re-emerged both in the wild deer population and also on cattle and captive cervid farms. Michigan lost its bovine TB accredited-free status in June 2000 and was required to adopt a testing program for all Michigan cattle, goats, bison, and captive cervids. In addition, other states could place movement restrictions on Michigan livestock at their discretion. Michigan agriculture is obviously concerned about disease-related costs and supports culling the deer population to eradicate the disease. However, such extreme measures could be costly, particularly since deer hunting is arguably the highest-valued use of the land in the infected region.

### **Wildlife management and disease control for Michigan white-tailed deer**

Bovine tuberculosis among Michigan white-tailed deer is primarily concentrated in a four-county area in the northeastern part of the lower peninsula, formally designated as deer management unit (DMU) 452 or less-formally as the 'core'. There is some limited infection beyond this area but the disease does not appear to be sustainable outside the core, leading many to speculate that the core exhibits unique features that have enabled the disease to become endemic (Hickling 2002).<sup>1</sup> These features include human-environment interactions, with supplemental feeding programs being a particular concern. Several hunt clubs in the core sponsor feeding programs that sometimes even dump tractor-trailer loads of food in the woods and fringe areas.<sup>2</sup> These

---

<sup>1</sup> Conventional wisdom held that the disease was not self-sustaining in wildlife populations (Hickling 2002). In fact, prior to 1995, only eight cases of bovine TB had ever been reported in wild deer from North America (Schmitt et al. 1997).

<sup>2</sup> The many hunt clubs in this area primarily exist to facilitate deer hunting. Originating in the late 1800's and early 1900's, these clubs purchased large amounts of land in the area for members from southern Michigan on which to hunt. This land was desirable for the clubs as it was easily accessible from highways and, as it consisted of generally poor soil for agronomic purposes, the land was inexpensive (Hickling 2002). The historic density of deer in the area is estimated to have been seven to nine deer per square kilometer (O'Brien et al. 2002). This low

massive piles of food can be seen from the air along with the tracks of thousands of congregating deer. There are economic reasons for providing this food, including increasing the carrying capacity of deer in the core. But such practices could also lead to increased transmission of the disease as deer congregate, and the supplementary food could also reduce the mortality rate of the disease by supporting sick animals.

### **A model of infectious disease**

Consider a wildlife (deer) population and a livestock (cattle) population that inhabit a particular land area. The deer population is free to roam while cattle are relegated to a number of farms (we consider the aggregate cattle population as opposed to farm-level populations for simplicity). Deer-cattle contact is possible in the absence of biosecurity investments to prevent this.

First consider the cattle population,  $x$ . This population grows naturally according to the net growth function  $g(x)$ . Farmers can add to or diminish their stocks through trade, with net sales denoted by  $y$ . Cattle become infected through contact with infected deer,  $z$ . Each infected deer makes on average  $\tilde{b}$  infectious contacts per cow in each time period, although these contacts can be reduced by investments,  $I$ , in biosecurity capital,  $K$ . The resulting disease prevalence rate is  $h = [\tilde{b}(1 - gK)]z$ , where  $\gamma$  is a parameter indicating biosecurity effectiveness. The total number of infected cows is  $\tilde{b}zx(1 - gK)$ . We assume on-farm testing allows farmers to identify and remove all infected cattle in each period. Given this specification, the cattle stock evolves according to

$$(1) \quad \dot{x} = g(x) - y - \tilde{b}zx(1 - gK).$$

---

carrying capacity was not conducive to easy hunting so the hunt clubs began aggressive deer feeding programs to encourage deer herd growth. The feeding programs were quite successful in increasing deer density with the density estimated at around 25 deer km<sup>2</sup> by the mid-1990's. As hunting is the highest valued use of land in the infected region, whole-sale changes to existing regulations and property-rights are not popular.

Now consider the aggregate deer population,  $N$ , which consists of two sub-populations: a healthy but susceptible stock, denoted by  $s$ , and an infected stock, denoted by  $z$ . In the absence of exploitation or disease, the susceptible stock grows according to the logistic growth function,  $rs(1-N/k)$ , where  $r$  is the intrinsic growth rate and  $k$  is the carrying capacity. Following Barlow (1991a), the density-dependent part of this equation,  $(1-N/k)$ , depends on the aggregate population because all deer compete for the same habitat. Following Horan and Wolf (2003), supplemental feeding, denoted  $f$ , reduces the density-dependent part of growth,  $(1-(N/k)(1-\tau f))$ , where  $\tau$  is a parameter. Growth is also reduced as members of the susceptible stock become infected, which occurs when a susceptible animal comes into contact with either an infected deer or an infected cow.<sup>3</sup> The  $z$  infected deer make on average  $\hat{b}(1+nf)z$  contacts with other deer, with only  $s/N$  contacts being with susceptible deer (assuming infected and susceptible deer are uniformly distributed across the land area), for a total of  $\hat{b}(1+nf)zs/N$  infected contacts in each time period.<sup>4</sup> Here,  $\hat{b}$  is the base contact rate and  $v$  is a parameter indicating the degree to which supplemental feeding increases contacts by bringing more deer together. Infected cows can also infect the susceptible deer herd. Each infected cow makes an average of  $\phi(1-\gamma K)$

---

<sup>3</sup> Direct contact with an infected animal is not required, although we model it this way for simplicity. Most infections on farms come from indirect contact, for instance consuming leftover food or water of infected animals.

<sup>4</sup> Disease transmission can be modeled in a number of ways. Historically, ecologists have used a mass action or density-dependent transmission function,  $\beta zs$ , where  $\beta$  is the contact rate per infectious deer – and this is how we have modeled deer-cow transmission because each population must be positive for cross-species transmission to occur. McCallum et al. (2001) note, however, that the mass action model often does not hold up empirically for within-species transmission. A competing transmission function, which we adopt for deer-deer transmission, is frequency-dependent or density-independent transmission. This form of transmission often fits data better than mass action for diseases such as cowpox in bank voles and wood mice (Begon et al. 1998, 1999) and brucellosis in Yellowstone bison (Dobson and Meagher 1996). Both mass action and frequency-dependent models assume infected and healthy wildlife are uniformly distributed across the landscape. McCallum et al. (2001) propose several alternative functional forms that may be more appropriate for non-uniformly distributed populations. One of these forms reduces to mass action at one extreme parameter value and frequency-dependent transmission at the other extreme, although the form is generally too complex to apply analytically. Still, there is no generally-accepted modeling approach and the data is too sparse in most cases to determine which form is appropriate (McCallum et

contacts per deer in each time period, for a total average of  $\varphi\eta xN(1-\gamma K)$  contacts (i.e.,  $\varphi(1-\gamma K)$  contacts times  $\eta xN$  infected cows).

The final activity affecting growth of the susceptible deer population is harvesting. Selective harvesting of infected deer may not be an option – it is often difficult to identify infected individuals prior to the kill because outward signs of an illness may not manifest (MDA 2002; Williams et al. 2002). Harvesting will therefore include both healthy and infected individuals, which could be costly as healthy deer are highly valued for recreational purposes.<sup>5</sup> Given non-selective harvesting, a manager can only choose the aggregate harvest,  $h$ , with the harvest from each stock depending on the proportion of animals in that stock relative to the aggregate population. That is,  $h_s = hs/N$  and  $h_z = hz/N$ , where  $h_i$  denotes the harvest from population  $i$ . Given this specification, the equation of motion for the susceptible stock is (Barlow 1991a; Heesterbeek and Roberts 1995)

$$(2) \quad \dot{s} = rs(1 - (N/k)(1 - tf)) - \hat{b}(1 + nf)zs/N - j\tilde{b}zxs(1 - gK)^2 - hs/N.$$

The infected stock also grows according to a logistic growth function (assuming infected mothers pass the disease to their young, either *in utero* or shortly after birth through contact; this would be common among birds and mammals), although the disease increases mortality by a rate of  $\alpha(1-\delta f)$ , where  $\alpha$  is the base mortality rate and  $\delta$  represents the extent to which supplemental feeding reduces disease mortality. The only other difference with (2) is that the infected stock increases when susceptible deer become infected. The equation of motion for the infected stock is (Barlow 1991a; Heesterbeek and Roberts 1995)

---

al.). For simplicity and because frequency-dependence fits the data better than mass action in many cases, we apply the frequency-dependent form.

$$(3) \quad \dot{z} = rz(1 - (N/k)(1 - tf)) - \mathbf{a}(1 - df)z + \hat{\mathbf{b}}(1 + nf)zs/N + \mathbf{j} \tilde{\mathbf{b}}zxs(1 - gK)^2 - hz/N.$$

It is more intuitive and convenient to work in terms of the variables  $N$  and  $\theta$  instead of  $s$  and  $z$ , where  $\theta$  represents the infected proportion of the population – the prevalence rate. The relations  $z = \theta N$  and  $s = (1 - \theta)N$  can be used to substitute for  $z$  and  $s$  in equations (1)-(3), and without loss we can instead focus on the following equations of motion

$$(4) \quad \dot{x} = g(x) - y - \tilde{\mathbf{b}}qNx(1 - gK),$$

$$(5) \quad \dot{N} = rN(1 - (N/k)(1 - tf)) - \mathbf{a}(1 - df)qN - h,$$

$$(6) \quad \dot{q} = [\hat{\mathbf{b}}(1 + nf) + \mathbf{j} \tilde{\mathbf{b}}x(1 - gK)^2 - \mathbf{a}(1 - df)](1 - q)q.$$

In the absence of supplemental feeding (i.e.,  $f=0$ ) and with no disease transmission from cows to deer, harvesting strategies alone cannot affect disease prevalence (McCallum et al. 2001): the disease dies out on its own when  $\hat{\mathbf{b}} < \mathbf{a}$ , and all animals become infected when  $\hat{\mathbf{b}} > \mathbf{a}$ . In the latter case, the disease can only be controlled by reducing  $\hat{\mathbf{b}}$  and/or increasing  $\alpha$ . With supplemental feeding (and without cattle-deer transmission), the disease *could* be endemic in the core if  $\beta(1 + \nu f) > \alpha(1 - \delta f)$ , and it would necessarily be endemic in this case if it were also true that  $r > \alpha$ , as is widely believed.<sup>6</sup> If  $\beta > \alpha$ , then the disease will persist regardless of feeding or hunting choices (apart from wildlife eradication). But if  $\beta < \alpha$ , then the disease would be eliminated by setting  $f < [\alpha - \beta]/[\beta\nu + \alpha\delta]$  for some time. The feeding rate must be even smaller if deer can contract the disease from cattle, i.e.,  $f < [\mathbf{a} + \hat{\mathbf{b}} - \mathbf{j} \tilde{\mathbf{b}}x(1 - gK)^2]/(d\mathbf{a} + n\hat{\mathbf{b}})$ , although

---

<sup>5</sup> Non-selectivity is not unique to the current situation. For instance, hunters/fishermen cannot selectively harvest from different cohorts within exploitable populations of many species (Reed 1980; Clark 1990), and by-catch of non-targeted species is often a problem in fisheries.

<sup>6</sup> The disease would not be sustainable outside the core if  $\beta_o < \alpha_o$ , where  $\beta_o$  and  $\alpha_o$  represent parameter values outside the core area and which may differ from  $\beta$  and  $\alpha$  due to human-environment interactions apart from feeding.



biosecurity investments would ease this requirement. A smaller  $f$  means the disease is eliminated sooner but at an interim cost of lost deer productivity.

### **An Economic Model**

The social planner evaluates net benefits among wildlife, agricultural, and other interests. We consider only the first two in this model. Wildlife managers have indicated two objectives when dealing with the disease: reduce the number of diseased animals and control the spread of the disease. To accomplish these goals, the choice variables under consideration are harvest levels and the amount of food provided by feeding programs (Hickling 2002).<sup>7</sup> However, choices made in the agricultural sector must also be considered, for economic damages to this sector depend not only on the wildlife management choices but also the on-farm responses to risks of livestock infection by wildlife.

Consider the hunting sector. Hunters gain utility from the actual process of shooting wildlife and/or consuming meat and other wildlife products. The (constant) marginal utility from harvesting healthy wildlife is denoted  $p$ , which is not less than the (constant) marginal utility from harvesting infected wildlife,  $p_z$ , i.e.,  $p \geq p_z$ . For simplicity and without loss, we set  $p_z = 0$  so that harvests of infected animals yield no benefits. The benefits from hunting are therefore  $phs/N = p(1-\theta)h$ . Clearly, greater disease prevalence damages the hunting sector in terms of foregone harvest benefits. Assume harvests occur according to the Schaefer harvest function (see Conrad and Clark 1987), and that the unit cost of effort,  $c$ , is constant. Then total harvesting costs, restricted on the *in situ* stocks, are  $(c/q)h/N$ , where  $q$  is the catchability coefficient. The unit cost of supplemental feed is  $w$ .

Now consider the agricultural sector. Cattle can be sold/purchased at a constant price of  $b$ . The cost of maintaining the herd is given by  $m(x)$  ( $m \leq 0$ ,  $m^2 > 0$ ). We assume infected cattle can be removed costlessly, but of course this reduction in the stock implies an opportunity cost for the farmer and hence livestock damages are endogenous. Finally, biosecurity investments  $I$  are made at a constant cost of  $u$ , with capital accumulating according to the equation of motion

$$(7) \quad \dot{K} = I - \zeta K,$$

where  $\zeta$  represents depreciation.

Given the discount rate  $\rho$ , an economically optimal allocation of harvests, feeding, biosecurity investments, and cattle stocking rates solves

$$(8) \quad \underset{h,f,I,x}{Max} SNB = \int_0^{\infty} [p(1-q)h - (c/q)(h/N) - wf + by - m(x) - uI] e^{-rt} dt,$$

subject to the equations of motion (4) – (6).<sup>8</sup> The current value Hamiltonian is

$$(9) \quad \begin{aligned} H = & p(1-q)h - (c/q)(h/N) - wf + by - m(x) - uI \\ & + I[rN(1 - (N/k)(1 - \theta)) - a(1 - d_f)qN - h] \\ & + \lambda[\hat{b}[1 + \eta f] + j \tilde{b}x[1 - gK]^2 - a[1 - d_f](1 - q)q] + y[I - \zeta K] \\ & + \pi[g(x) - y - \tilde{b}qNx(1 - gK)] \end{aligned}$$

where  $\lambda$ ,  $\phi$ ,  $\psi$ , and  $\pi$  are the co-state variables associated with  $N$ ,  $\theta$ ,  $K$ , and  $x$ , respectively.

The marginal impact of net cattle sales on the Hamiltonian is

$$(10) \quad \partial H / \partial y = b - p.$$

<sup>7</sup> Michigan announced a goal of eradicating the disease by 2010. To that end, the wild white-tailed deer population in the area was to be decreased through hunting programs that sold increased licenses. In addition, the practice of legally feeding deer in the infected area was ended and the practice of baiting was temporarily ended.

<sup>8</sup> It is implicitly assumed that  $h, f, I \geq 0$ , that  $f \leq \min(1/\delta, 1/\tau)$ , and that  $K \leq 1/\gamma$ . A value of  $f > 1/\delta$  would result in a negative mortality rate due to the disease, which is not possible. A value of  $f > 1/\tau$  would result in a negative density

Net cattle sales should be as large as possible when  $\partial H/\partial y > 0$ , and they should be as small as possible when  $\partial H/\partial y < 0$ . A singular path should be followed whenever  $\partial H/\partial y = 0$ . In this latter case  $\dot{p} = 0$  since  $b$  is fixed.

The marginal impact of biosecurity investments on the Hamiltonian is given by

$$(11) \quad \partial H / \partial I = -u + y .$$

If this expression is positive so that the marginal value of capital exceeds the marginal cost of investment, then investments should be set at their maximum levels. If this expression is negative then  $I=0$  is optimal. The singular solution is pursued when marginal investment costs and the marginal value of capital are equated. In this case,  $\dot{y} = 0$  since  $u$  is fixed.

The marginal impact of harvests on the Hamiltonian is given by

$$(12) \quad \partial H / \partial h = p(1 - q) - c/(qN) - I .$$

If this expression is positive so that marginal rents exceed the marginal user cost, then harvests should be set at their maximum levels. If this expression is negative then no harvesting should occur. The singular solution is pursued when marginal rents and the marginal user cost are equated.

Now consider the marginal impacts of feeding on the Hamiltonian

$$(13) \quad \partial H / \partial f = -w + I[r(N^2/k)t + adqN] + f[\hat{b}u + ad](1 - q)q .$$

---

dependence factor, which also does not seem realistic. Finally, a value of  $K > 1/\gamma$  would result in negative disease transmission. In our numerical example these assumptions are explicit.

Feeding can be thought of as an investment in both the productivity of the resource and of the disease. As we show below, the solution has similarities but also important differences than when investments are made in harvesting capital (see Clark et al. 1979). The singular solution should be followed whenever the unit cost of feeding equals the *in situ* net marginal value of feeding on the two state variables. The *in situ* net marginal value is the difference between the marginal benefits of feeding on the overall stock (which includes increased productivity and decreased mortality) and the marginal costs of feeding in terms of an increased proportion of infected animals (due to increased transmission and decreased mortality among the infected stock). If the marginal *in situ* values exceed the unit cost, then feeding should proceed at some maximum rate. If the unit cost exceeds the *in situ* value then feeding should optimally cease.

The necessary arbitrage conditions for an optimal solution are given by

$$(14) \quad \dot{I} = rI - \partial H / \partial N = rI - ch / (qN^2) - I[r - 2r(N/k)(1 - tf) - a(1 - df)q] + pq\tilde{b}_x(1 - gK),$$

$$(15) \quad \dot{f} = rf - \partial H / \partial q = rf + ph + I a(1 - df)N - f[b(1 + uf) + j\tilde{b}_x(1 - gK)^2 - a(1 - df)](1 - 2q) + pN\tilde{b}_x(1 - gK),$$

$$(16) \quad \dot{y} = ry + f[2j\tilde{b}_xg(1 - gK)](1 - q)q + yz - p\tilde{b}qN_xg,$$

$$(17) \quad \dot{p} = rp + m'(x) - f[j\tilde{b}(1 - gK)^2](1 - q)q - pg'(x) + p[\tilde{b}qN(1 - gK)].$$

### *The Multi-Singular Solution*

Consider harvesting, feeding, net sales, and investment choices along a singular path, so that conditions (10) – (13) all vanish. We refer to such a path, in which the solution is singular for multiple controls, as a multi-singular path (solutions that are singular for only one control variable might also be possible, and sometimes these are the only feasible singular possibilities,

e.g., see Clark et al. 1979). Differentiating condition (12) with respect to time, substituting the right-hand-side (RHS) of condition (14) in for  $\dot{I}$ , and using (10) and (12) to substitute for the co-state variables  $\lambda$  and  $\pi$ , we have the expression

$$(18) \quad \mathbf{r} = r - \frac{2rN}{k}(1 - \mathbf{t}f) + \frac{[c/(qN^2)][rN[1 - (N/k)(1 - \mathbf{t}f)]] - b\mathbf{q}\tilde{\mathbf{b}}x(1 - \mathbf{g}K)}{p(1 - \mathbf{q}) - c/(qN)} \\ - \frac{p[\mathbf{b}(1 + \mathbf{u}f) + \mathbf{j}\tilde{\mathbf{b}}x(1 - \mathbf{g}K)^2](1 - \mathbf{q})\mathbf{q}}{p(1 - \mathbf{q}) - c/(qN)}$$

Equation (18) is a variant of the conventional “golden rule” for renewable resource management: the rate of return for holding the healthy stock *in situ* equals the marginal productivity of the stock, plus net marginal stock effects (i.e., the marginal cost savings that accrue as harvests come from a larger stock minus the marginal damages in terms of increased cattle disease, normalized by marginal user cost), minus the (normalized) value of foregone revenues as some of the remaining healthy *in situ* stock will become infected and result in a larger proportion of infected deer in future harvests.

Equation (18) must hold at all times along the multi-singular path. In conventional autonomous renewable resource models, the singular path is a single point,  $N^*$ , because the golden rule is only a function of the stock and can be solved for a unique value of  $N$ . In contrast, condition (18) is a function of one of the control variables,  $f$ . Solving (18) for  $f$  as a function of the current state variables, the result is a nonlinear feedback law along a multi-dimensional singular arc, i.e.,  $f=f(N, \theta, K, x)$  (Bryson and Ho 1975). As we describe below, this feedback rule results in the multi-singular solution being a path and not simply a steady state point.

Now differentiate condition (13) with respect to time, substitute the right-hand-side (RHS) of condition (15) in for  $\dot{F}$ , and the RHS of (14) in for  $\dot{I}$ . Using (10), (12) and (13) to substitute for the co-state variables  $\pi$ ,  $\lambda$ , and  $\phi$ , we get a golden rule expression for managing the

prevalence rate of infected wildlife. The explicit form of this expression is too complex to present here, but in implicit form it is written

$$(19) \quad \mathbf{r} = F(N, \mathbf{q}, K, x, h, f).$$

Equation (19) depends on two control variables,  $h$  and  $f$ . This equation must also hold along the multi-singular path. If we plug the feedback law for  $f$  into this expression, it is possible to construct a feedback law for the harvest,  $h=h(N, \theta, K, x)$ .

Along the multi-singular path, equations (10) and (11) imply that  $\dot{\mathbf{p}} = \dot{\mathbf{y}} = 0$ . Using this result in equations (16) and (17), it is possible to solve for  $K(N, \theta)$  and  $x(N, \theta)$  (see Appendix B for a discussion of the bounds of  $K$ ). Plugging these relations back into the feedback laws for  $h$  and  $f$ , we have  $h(N, \theta)$  and  $f(N, \theta)$ . The controls for investment and net cattle purchases are then  $I(N, \mathbf{q}) = \dot{K}(N, \mathbf{q}) + \mathbf{d}K(N, \mathbf{q})$  and  $y(N, \mathbf{q}) = g(x(N, \mathbf{q})) - \tilde{\mathbf{b}}\mathbf{q}Nx(N, \mathbf{q})[1 - \mathbf{g}K(N, \mathbf{q})] - \dot{x}(N, \mathbf{q})$ . Finally, the feedback laws  $h(N, \theta)$  and  $f(N, \theta)$  can be plugged into the differential equations (7) and (8) to solve for the optimal path along the singular arc.

Because the singular arc is two-dimensional, the entire  $(N, \theta)$  plane – or at least a subset of it – satisfies the necessary conditions for the multi-singular solution. Indeed, assuming  $f(N_0, \mathbf{q}_0) > 0$  and  $h(N_0, \mathbf{q}_0) > 0$ , the multi-singular path can generally be found numerically by using the nonlinear feedback laws along with the equations of motion (7) and (8) and the initial states  $N_0$  and  $\mathbf{q}_0$ . Constraints on the controls may provide some additional restrictions that limit the space over which a multi-singular solution may arise. We explore these constraints as we discuss the numerical model below.

### *Numerical example*

We examine the optimal solution numerically because the feedback rules and the differential equations that define the solution are too complex to analyze analytically. The data used to parameterize the model are described in Appendix A. While we have made every effort to calibrate the model realistically, research on the Michigan bovine TB problem is still at an early stage so knowledge of many parameters is limited. The following analysis is therefore best viewed as a numerical example.

The numerical solution is presented in Figure 1 for the case of  $\rho=0.05$ . Although not presented, the only interior equilibrium point is an unstable focus that is not to be pursued. Instead, we find an interior cycle is optimal.<sup>9</sup> Horan and Wolf (2003) also found an interior cycle to be optimal, although there are important qualitative differences between those results and the present results. The differences stem from the fact that Horan and Wolf (2003) modeled marginal damages to the cattle sector to be constant, with the management of the cattle sector not explicitly considered.

We now describe the optimal numerical solution. The first result is that biosecurity capital should optimally be set at a rate of zero for any interior combination of  $N$  and  $\theta$ . The reason is that the cattle industry in this region is not highly profitable, while biosecurity investments are costly and not very effective at the margin. The same result holds even if we reduce the investment cost  $u$  by several orders of magnitude. This also implies that cattle and deer can interact, infecting each other in the absence of deer fencing.

---

<sup>9</sup> Note that  $N=0$  is not an optimal steady state because the marginal cost of exterminating the wildlife population becomes very large while the marginal benefits of extermination approach zero. Equilibria involving  $\theta=0$  are not optimal either because it takes too long for the disease to die out naturally.

Given that  $K=0$  and given  $N_0$  and  $\theta_0$ , as represented by point  $A$  in Figure 1, the multi-singular path  $I$  is initially followed. Along this path both the deer stock and the prevalence rate increase due to increased supplemental feeding. The result that feeding should be initially encouraged runs contrary to Michigan's current policy approach of banning feeding. Feeding represents an investment in stock productivity, initially increasing the stock while enabling large harvests. Although the disease prevalence rate increases along the singular path, the increased damages are offset by the rewards of larger near-term harvests. Damages – in terms of transmission from deer to cattle and cattle back to deer – are also mitigated by diminishing the cattle herd along this path until the  $x=0$  curve is reached, at which point the cattle stock is optimally depleted. The cattle stock remains depleted at all points to the right of the  $x=0$  curve. This result is in stark contrast to the more conventional policy recommendation of eradicating all wildlife in order to protect livestock. The reason is that deer are highly valued in this region while the cattle sector is marginally productive. Selling off all cattle in the region yields near-term gains while simultaneously eliminating damages to the cattle sector.

Once the  $x=0$  curve is reached, feeding and also deer prevalence rates continue to grow along the path  $I$ . Eventually  $f(N,\theta) = f^{\max} = 10,000$ , represented by the boundary  $f = f^{\max}$  in Figure 1. The prevalence rate at this point is approximately four times larger than in the Horan and Wolf (2003) solution. The larger rate in the present model arises because marginal damages to the cattle sector are zero when the  $f = f^{\max}$  boundary is reached, while marginal damages to the cattle industry were assumed to be positive and constant in the Horan and Wolf model.

The  $f = f^{\max}$  boundary creates a blocked interval that prevents the state variables from following the multi-singular path (Arrow 1964; Clark 1990, p. 56). The feedback solution is myopic, but the farsighted planner knows the boundary is approaching. So the singular path is



abandoned (at least for the feeding control variable) prior to reaching the  $f = f^{\max}$  boundary, for instance at the point  $B$ , and an extremal value of  $f$  is chosen (Arrow 1964). Clark (1990, p.57) refers to this result as the “premature switching principle”.

At the instant at which the multi-singular path  $I$  is abandoned, say time  $T$ , it becomes optimal to pursue the singular solution for  $N$  conditional on  $K=0$ ,  $x=0$ , and conditional on the extremal value of  $f$  (note there are two possible extremal values for  $f$ :  $f^{\max}$  and 0). This singular path is characterized by equation (18), holding  $f$ ,  $K$ , and  $x$  fixed at their constrained values. Given these exogenous constraints, equation (18) can be solved for  $N(\theta)$ , with  $\theta$  moving exogenously through time as a function of the constrained variables. The result is a singular path for  $N$ , referred to as the constrained singular path, which is essentially a non-autonomous singular path given the exogenous movement of  $\theta$  (see Clark 1990 or Conrad and Clark 1987 for examples of non-autonomous singular paths) and is optimally approached along a most rapid approach path (MRAP).

As discussed in Horan and Wolf (2003), it is not optimal to feed at the extremal value  $f^{\max}$  along the constrained singular path. If we were to set  $f = f^{\max}$ , the corresponding deer stock levels would lie to the left of the  $f=0$  curve. But in this region of the phase plane the necessary conditions imply that feeding is optimally zero, and so  $f = f^{\max}$  cannot be optimal. Rather, it is optimal to set  $f=0$  and pursue the conditionally optimal path based on this value – which happens to coincide with the  $f=0$  curve. So at time  $T$  it is optimal to cull the deer herd (represented by path 2) to point  $C$  – the population that arises at the  $f=0$  curve for the current value of  $\theta$ . The  $f=0$  curve is then followed (path 3).

Disease prevalence diminishes while wildlife stocks increase along the conditionally singular path 3, until point  $D$  is reached. An analogous outcome arises in Horan and Wolf’s

solution, although the process takes much longer in the present model because point  $C$  will be associated at a much higher prevalence rate in the present model. Prior to reaching point  $D$ ,  $\theta$  becomes small enough that it becomes optimal to raise cattle again, and so society is again willing to incur some small damages to the cattle industry.<sup>10</sup>

Although continuation along the constrained singular path 3 would eventually lead to a disease-free deer stock (after which time feeding could be reintroduced without creating any disease problems), that outcome is not pursued because the opportunity cost of waiting for the disease to die out is too high relative to the gains that can be made from re-investing in deer productivity. The marginal productivity impact of supplemental feeding depends on the size of the deer population. If the deer stock is relatively small, such as at point  $C$ , then feeding is costly: it results in only a small productivity boost while simultaneously causing increased disease prevalence. But when point  $D$  (approximately  $N=6,500$  and  $\theta=0.02$ : both of which are larger than in Horan and Wolf's baseline solution) is reached, feeding again becomes beneficial: small amounts of supplemental feeding can have a significant productivity boost while adding little to disease prevalence. This is reflected by the relatively flat slope of path 4 in the vicinity of  $D$ . Once on path 4, a similar process of feeding and culling continues and a cycle emerges along paths 4-2-3. To simplify the graphical presentation, we have drawn the initial and subsequent deer culls occurring along path 2, although it is more likely that the initial cull will occur along a different path than the others.<sup>11</sup> In any case, the disease is never eradicated

---

<sup>10</sup> Of course, we are not assuming any fixed costs associated with re-starting the cattle industry. If there are large fixed costs associated with this, then society might want to wait longer before re-starting the industry – or it may never re-start the industry.

<sup>11</sup> To fully and accurately characterize the solution it is necessary to find the optimal time  $T$  at which path 1 is abandoned and the optimal times at which path 4 is abandoned. This is beyond the scope of the current paper.

because the deer are highly valuable and feeding intermittently becomes a good investment to boost the productivity of the deer stock.

In many respects the optimal path is similar to that of Clark et al. (1979), who analyze irreversible investments in harvesting capacity for renewable resources. They find it is optimal to temporarily over-capitalize (relative to the steady state) prior to a stock-depletion phase. The reason is that the larger capital levels allow more harvesting early on, which generate greater near-term benefits prior to advancing to the steady state. Somewhat analogously in our model, we find that initial and intermittent future investments in resource productivity create opportunities for near-term gains. An important difference between our model and Clark et al.'s model is that a steady state is not optimal in our model. Unlike Clark et al., investment in our model (via feeding) produces adverse effects on resource dynamics: along with the productivity enhancing investments comes the unwanted side-effect of the disease, and sustained investment (feeding) would only lead to increasing disease prevalence. If allowed to continue unabated, this increasing prevalence eventually causes damages (to deer hunters) to swamp benefits. Therefore, intermittent dis-investment in the disease is warranted.

## **Conclusions**

In this paper, we investigated the economics of disease control in interactive wildlife-livestock populations, expanding upon prior work by explicitly taking livestock management choices into account. From our numerical example of bovine tuberculosis in Michigan deer populations, we found that the ability to mitigate damages via changes in on-farm choices results in greater disease prevalence rates in deer and a smaller likelihood that eradication of deer will be an optimal strategy. This is reasonable since the ability to mitigate damages reduces the opportunity cost of allowing larger disease prevalence rates in deer. Perhaps surprising,

however, is that we found it optimal to remove the cattle industry in the infected area. The reason is that the cattle industry in the infected area is only marginally profitable, and shutting it down reduces marginal damages to the cattle industry to zero. Without having to account for these marginal damages, deer can be managed at larger population levels and larger prevalence rates to support the highly-valued recreational hunting sector. But still there are incentives to control the disease due to the reduced productivity of the deer herd and the reduced hunting values that emerge as the deer prevalence rate increases.

In any case, we find that eradication of the disease is not likely to be economically optimal. It takes too long for the disease to dissipate naturally once supplemental feeding is halted, which is not surprising considering that it took sixty-two years to eliminate the disease in cattle herds under much more controlled conditions. It is also too difficult and costly to kill all the deer in the infected area, as managers in Michigan are currently discovering. Instead, it is optimal for the disease to remain endemic in the area at very low levels, with intermittent investments (via supplemental feeding) in *in situ* deer productivity. Of course an endemic disease is not always optimal. If marginal damages, feeding costs and/or disease mortality are large enough, we find that it may be optimal to delay feeding-induced productivity enhancements and in favor of disease eradication.

Although the model was applied to the specific case of bovine TB in deer herds, the model and results are likely to be applicable to other wildlife disease problems – even those problems where supplemental feeding is not an issue. Supplemental feeding decisions in our model represent the easiest method of controlling disease transmission for the Michigan case, and the control of disease transmissions would likely be a part of any wildlife disease management strategy. For other diseases, alternative environmental variables could be

manipulated in ways that reduce disease transmission, and it is reasonable to believe that such actions might result in tradeoffs in *in situ* productivity (e.g., if contact is somehow reduced then fertility might also be expected to decline). Hence the current model provides a foundation for analyzing a range of wildlife disease problems.

Finally, an important caveat to our results is that the disease was assumed to be unsustainable beyond the core area. This is reasonable for the Michigan bovine TB problem, but it may not be the case for some other diseases. Rather, it might be possible for some other diseases to spread among additional populations. Such a situation might imply greater marginal damages due to the disease and hence more incentives to contain the disease. Additional tradeoffs may also arise involving the management of spatially differentiated populations that possibly interact through migratory processes. A spatially explicit analysis would be required in such instances to fully assess the implications of spatial disease transmission.

## **Appendix A. Model Calibration**

The model is calibrated using parameters obtained from a variety of sources. The following parameter values were derived in Horan and Wolf (2003) using the results of the listed sources:  $N_0 = 13,298$  (Hill 2002);  $\theta_0 = 0.023$  (O'Brien et al. 2002; Hickling 2002);  $k = 14,049$  (Miller et al. 2003; O'Brien et al. 2002),  $\tau = 0.00008$  (Miller et al. 2003; O'Brien et al. 2002; Hickling 2002);  $\nu = 2.64 \times 10^{-6}$  and  $\beta = 0.339$  (Miller and Corso's 1999; McCarty and Miller 1998; Miller et al. 2003);  $r = 0.5703$  (Rondeau and Conrad 2003);  $\alpha = 0.356$  and  $\delta = 5.34 \times 10^{-5}$  (Hill 2002);  $p = \$1270.80$  (Boyle et al 1998; Frawley 1999; and U.S. DOI-FWS 1996);  $c/q = \$231,192$  (Rondeau and Conrad 2003);  $w = 36.53$  (Miller et al. 2003; anecdotal evidence).

Additional parameters are required for the present model. We adopt Bicknell et al.'s value of an intrinsic growth rate for cattle of 0.67. The cattle carrying capacity is taken to be 0.35 head/acre, based on observations for the core. There are approximately 80 farms in the core area, with an average size of 100 acres (USDA 1996). The average price per cow is assumed to be \$1000, and the maintenance cost per cow is taken to be \$260 (which includes a base maintenance cost of \$400/ beef cow and \$300 net profit per dairy cow, assuming the proportions remain constant). Assuming each farm is of average size and square shape, the cost of installing a deer fence is approximately \$7/linear foot, which amounts to \$58,436 per farm. Capital is measured as the number of farms fenced in, so the maximum value of  $K$  is taken to be 80, which implies that  $\gamma=1/80$ . Finally, the deer-to-cattle and cattle-to-deer transmission coefficients are  $\tilde{b} = j = 0.0003$  (USDA 1996).

## Appendix B. Bounds on Capital Accumulation

To investigate investment choices and the impact on deer and cattle management, consider the singular solution for capital investment. As described above, condition (11) implies that  $\dot{\psi}=u$  and  $\dot{y}=0$  along this singular path. Plugging this result into equation (16) (and also assuming a singular solution for  $y$ ), we can solve for the following value of  $K$ , which must hold for all time along the singular path:

$$(B1) \quad K = \frac{1}{g} + \frac{b\tilde{b}qNxg - (r+z)u}{|g^2f2j\tilde{b}x(1-q)q|}$$

Implicit in our model is that cross-species disease transmission ceases for all values of  $K \geq 1/\gamma$ ; there are no incentives for investing such that  $K > 1/\gamma$ , since  $K = 1/\gamma$  yields the same effect at a lower cost. The first RHS term in (B1),  $1/\gamma$ , therefore represents the upper bound on capital

accumulation. Clearly,  $K < 1/\gamma$  if the second RHS term is negative, and  $K = 1/\gamma$  if the second RHS term is positive.

Consider the second RHS term in (B1). The numerator,  $\Delta = b\tilde{b}qNxg - (r+z)u$ , is the value of the reduction in deer-to-cattle transmission due to a marginal increase in capital ( $b\tilde{b}qNxg$ ) less the opportunity cost of investment ( $(r+z)u$ ). The denominator,  $\Gamma = |g^2f^2j\tilde{b}x(1-q)q|$ , is the value of the reduction in cattle-to-deer transmission due to a marginal increase in capital, evaluated when  $K=0$ . If  $\Delta < 0$ , then capital should be set at a positive level only if  $\Gamma > 0$ ; otherwise  $K=0$  is optimal. Assuming  $\Gamma > 0$ , the  $\Delta < 0$  outcome depends on the product of the current values of the state variables  $N$ ,  $\theta$ , and  $x$ . If either of these variables is small, then the risk of transmission is sufficiently low that complete biosecurity protection (i.e.,  $K=1/\gamma$ ) is not supported on economic grounds. If  $\Delta \geq 0$  (e.g., if  $N$ ,  $\theta$ , and/or  $x$  were sufficiently large), then capital should be set at its maximum level regardless of the level of  $\Gamma$ . Note that a corner solution must arise if  $\Gamma=0$  (e.g., if  $\varphi=0$  so that there is no risk of cattle-to-deer transmission), with  $K=0$  if  $\Delta < 0$  and  $K=1/\gamma$  if  $\Delta > 0$ .

Assuming  $\Gamma > 0$ , then  $\Delta=0$  defines an iso-plane that divides  $(N, \theta, x)$  space into regions of risk and no-risk. Deer and cattle stocks would be managed simultaneously in the risk region. But in the no-risk region, conditions (14) and (15) clearly become independent from (16). This means that the deer and cattle stocks would optimally be managed separately, greatly simplifying the analysis. The deer and the deer prevalence rate would be managed in a manner similar to that described in the numerical analysis of the main text for the case in which  $x=0$ . Within the no-risk region, the cattle population would be managed according to the following golden rule, derived from (10) and (17)

$$(B2) \quad r = g'(x) - m'(x)/b$$

Equation (B2) can be solved for the singular value  $x^*$ , with net sales  $y$  being adjusted to achieve this level along a most rapid approach path (MRAP). Finally, investment will be maintained at the rate  $I=1/\delta$ , in order to offset depreciation, as long as the system remains in the no-risk region. However, capital would optimally be allowed to depreciate if the system were to move back into the no-risk region. This is a strong possibility with the deer population and prevalence rates cycling between high and low values.

## References

- Arrow, K.J. "Optimal Capital Policy, the Cost of Capital, and Myopic Decision Rules" *Annals of the Institute of Statistical Mathematics* 16 (1964): 21-30.
- Barlow, N. "A Spatially Aggregated Disease/Host Model for Bovine Tb in New Zealand Possum Populations" *Journal of Applied Ecology* 28(1991a): 777-793.
- Barlow, N. "Control of Endemic Bovine Tb in New Zealand Possum Populations: Results from a Simple Model." *Journal of Applied Ecology* 28(1991b): 794-809.
- Bicknell, K.B., J.E. Wilen, and R.E. Howitt. "Public Policy and Private Incentives for Livestock Disease Control." *Australian Journal of Agricultural and Resource Economics* 43(1999): 501-521.
- Boyle, K.J., B. Roach, and D. Waddington. *1996 Net Economic Values for Bass, Trout, and Walleye Fishing, Deer, Elk and Moose Hunting, and Wildlife Watching: Addendum to the 1996 National Survey of Fishing, Hunting and Wildlife Associated Recreation*, U.S. Fish and Wildlife Service, Washington DC, August 1998.
- Bryson, A.E. Jr., Ho, Y.C. *Applied Optimal Control: Optimization, Estimation, and Control*, Hemisphere Publishing, New York, 1975.
- Clark, C.W. *Mathematical Bioeconomics*, New York: Wiley, 1976.
- Clark, C.W., F.H. Clarke, and G.R. Munro. "The Optimal Exploitation of Renewable Resource Stocks: Problems of Irreversible Investment" *Econometrica* 47(1979): 25-47.
- Conrad, J.W. and C.W. Clark. *Natural Resource Economics: Notes and Problems*, New York: Cambridge University Press, 1987.



Dietrich, R.A., S.H. Amosson, and R.P. Crawford. "Bovine Brucellosis Programs: An Economic/Epidemiological Analysis." *Canadian Journal of Agricultural Economics* 35(1987): 127-140.

Ebel, E.D., R.H. Hornbaker, and C.H. Nelson. "Welfare Effects of the National Pseudorabies Eradication Program." *American Journal of Agricultural Economics* 74(1991): 638-645.

Frawley, B.J. 1999 Deer Harvest for Early Archery, Early Firearm, and Regular Firearm Deer Seasons in the Northeast Lower Penninsula. Michigan Department of Natural Resources, Wildlife Bureau Report Number 3304, December 1999.

Heesterbeek, J.A.P. and M.G. Roberts. "Mathematical Models for Microparasites of Wildlife," in *Ecology of Infectious Diseases in Natural Populations* (B.T.Grenfell and A.P. Dobson, eds.), Cambridge University Press, Cambridge, 1995.

Hicking, G. J. Dynamics of bovine tuberculosis in wild white-tailed deer in Michigan, Michigan Department of Natural Resources Wildlife Division Report No. 3363. March 2002.

Hill, H.R. The 2002 Deer Pellet Group Surveys, Michigan Department of Natural Resources, Wildlife Report No. 3376, August 2002.

Horan, R.D., and C.A. Wolf, "The Economics of Managing Wildlife Disease: Bovine TB in Michigan Deer Populations.", Selected paper, the annual meetings of the American Agricultural Economics Association, Montreal, CN, July 27-30, 2003.

Liu, C. "An Economic Impact Evaluation of Government Programs: The Case of Brucellosis Control in the United States." *Southern Journal of Agricultural Economics* 11(1979): 163-168.

Mahul, O. and A. Gohin. "Irreversible Decision Making in Contagious Animal Disease Control Under Uncertainty: An Illustration Using FMD in Brittany." *European Review of Agricultural Economics* 26(1999): 39-58.

McCarty, C.W. and M.W. Miller. "A Versatile Model of Disease Transmission Applied to Forecasting Bovine Tuberculosis Dynamics in White-Tailed Deer Populations."

McInerney, J. "Old Economics for New Problems – Livestock Disease: Presidential Address." *Journal of Agricultural Economics* 47(1996): 295-314.

Meagher, M. and M.E. Meyer. "On the Origin of Brucellosis in Bison of Yellowstone National Park: A Review." *Conservation Biology* 8(1994): 645-653.

Michigan Department of Agriculture (MDA), Michigan Bovine TB, <http://www.bovinetb.com> (downloaded August 21, 2002).

Miller, M.W. and B. Corso. *Risks Associated with M. bovis in Michigan Free-Ranging White-Tailed Deer: An Update to the 1996 Report*, U.S. Department of Agriculture, Animal and Plant

Health Inspection Service, Centers for Epidemiology and Animal Health, Fort Collins, November 1999.

Miller, R., J.B. Kaneene, S.D. Fitzgerald, and S.M. Schmitt. "Evaluation of the Influence of Supplemental Feeding of White-Tailed Deer (*Odocoileus Virginianus*) on the Prevalence of Bovine Tuberculosis in the Michigan Wild Deer Population." *Journal of Wildlife Diseases* 39: 84-95, 2003.

O'Brien, D.J., S.M. Schmitt, J.S. Fierke, S.A. Hogle, S.R. Winterstein, T.M. Cooley, W.E. Moritz, K.L. Diegel, S.D. Fitzgerald, D.E. Berrry, J.B. Kaneene. "Epidemiology of *Mycobacterium bovis* in Free-Ranging White-Tailed Deer in Michigan." *Preventive Veterinary Medicine* 54: 47-63, 2002.

Peterson, M.J. "Wildlife Parasitism, Science, and Management Policy." *Journal of Wildlife Management* 55(1991): 782-789.

Reed, W.J. "Optimum Age-Specific Harvesting in a Nonlinear Population Model." *Biometrics* 36 (1980): 579-593.

Rondeau, D. and J. Conrad. "Managing Urban Deer." *American Journal of Agricultural Economics* 85: 266-281, 2003.

Schmitt, S.M., S.D. Fitzgerald, T.M. Cooley, C.S. Bruning-Fann, L. Sullivan, D. Perry, T. Carlson, R.B. Minnis, J.B. Payeur, and J. Sikarskie. "Bovine tuberculosis in free-ranging white-tailed deer in Michigan." *Journal of Wildlife Diseases* 33(1997):749-758.

Simonetti, J.A. "Wildlife Conservation Outside Parks is a Disease-Mediated Task." *Conservation Biology* 9(1995): 454-456.

U.S. Department of Agriculture, Animal and Plant Health Inspection Service (USDA-APHIS). Foot-and-Mouth Disease, <http://www.aphis.usda.gov/oa/pubs/fsfmd301.htm>, January 2002 (downloaded August 30 2002).

U.S. Department of Agriculture, Animal and Plant Health Inspection Service (USDA-APHIS), "Appendix C: Simulation Model of Tuberculosis Risk to Cattle from Infection in Free-Ranging White-Tailed Deer", *Assessing the Risks Associated with M. Bovis in Michigan Free-Ranging White-Tailed Deer*. Center for Animal Disease Information and Analysis (CADIA) Technical Report No. 01-96.

U.S. Department of the Interior, Fisheries and Wildlife Service, U.S. Department of Commerce, Bureau of the Census. *1996 National Survey of Fishing, Hunting, and Wildlife-Associated Recreation*, Washington, DC. 1996.

Williams, E.S., M.W. Miller, T.J. Kreeger, R.H. Kahn, and E.T. Thorne. "Chronic Wasting Disease of Deer and Elk: A Review with Recommendations for Management." *Journal of Wildlife Management* 66(2002): 551-563.

Wolf, C.A. and J.N. Ferris. Economic Consequences of Bovine Tuberculosis for Michigan Livestock Agriculture, A report to the Michigan Department of Agriculture, 2000.

Wolfe, L.L., M.M. Conner, T.H. Baker, V.J. Dreitz, K.P. Burnham, E.S. Williams, N.T. Hobbs, and M.W. Miller. "Evaluation of Antemortem Sample to Estimate Chronic Wasting Disease Prevalence in Free-Ranging Mule Deer." *Journal of Wildlife Management* 66(2002): 564-573.

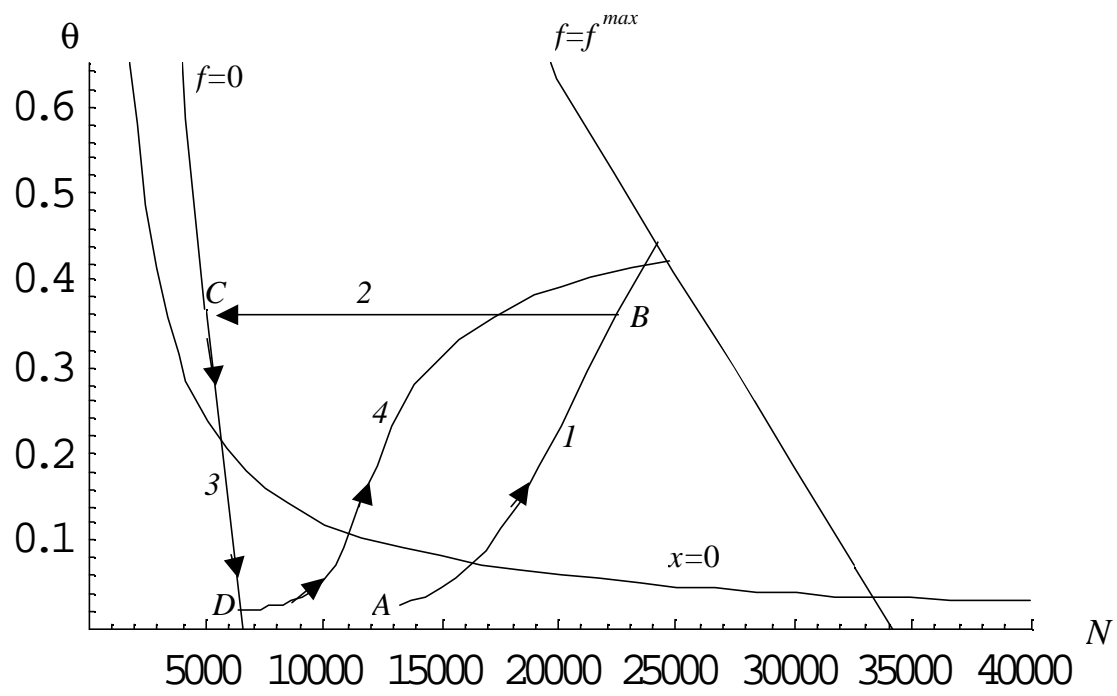


Figure 1. Solution of the numerical example